

AMENDMENTS TO THE CLAIMS:

1. (Original) A method for diagnosing a cancer in a mammal, comprising:
 - a) determining PKN gene copy number in a test sample from a region of the mammal that is suspected to be precancerous or cancerous, thereby generating data for a test gene copy number; and
 - b) comparing the test gene copy number to data for a control gene copy number, wherein an amplification of the gene in the test sample relative to the control indicates the presence of a precancerous lesion or a cancer in the mammal.
2. (Original) The method according to claim 1, wherein the cancer is a breast cancer, a colon cancer, an esophagus cancer, a bladder cancer, a brain cancer, a head and neck cancer, a kidney cancer, a liver cancer, a lymphoma cancer, a melanoma cancer, a pancreatic cancer, a lung cancer, an ovarian cancer, or a stomach cancer.
3. (Currently amended) A method for inhibiting cancer or precancerous growth in a mammalian tissue, comprising contacting the tissue with an inhibitor that interacts with **PKN protein**, PKN DNA or RNA and thereby inhibits PKN **[[gene]]** function.
4. (Original) The method according to claim 3, wherein the tissue is a breast tissue, a colon tissue, an esophageal tissue, a bladder tissue, a brain tissue, a head and neck tissue, a kidney tissue, a liver tissue, a lymphoma tissue, a melanoma tissue, a pancreatic tissue, a lung tissue, an ovarian tissue, or a stomach tissue.
5. (Original) The method according to claim 3, wherein the inhibitor is a siRNA, miRNA, an antisense RNA, an antisense DNA, a decoy molecule, or a decoy DNA.
6. (Original) The method according to claim 3, wherein the inhibitor contains nucleotides, and wherein the inhibitor comprises less than about 100 bps in length.
7. (Original) The method according to claim 3, wherein the inhibitor is a ribozyme.

8. (Original) The method according to claim 3, wherein the inhibitor is a small molecule.

9-10 (Canceled).

11. (Currently amended) A method for diagnosing a cancer in a mammal, comprising:

- a) determining the level of PKN in a test sample from a region of the mammal that is suspected to be precancerous or cancerous, thereby generating data for a test level; and
- b) comparing the test level of PKN to data for a control level, wherein an elevated test level of PKN of the test sample relative to the control level indicates the presence of a precancerous lesion or a cancer in the mammal.

12. (Original) The method according to claim 11, wherein the control level is obtained from a database of PKN levels detected in a control sample.

13. (Original) A method of blocking *in vivo* expression of a gene by administering a vector encoding PKN siRNA.

14. (Original) The method of claim 13, wherein the siRNA interferes with PKN activity.

15. (Original) The method of claim 13, wherein the siRNA causes post-transcriptional silencing of PKN gene in a mammalian cell.

16. (Original) The method of claim 15, wherein the cell is a human cell.

17. (Original) A method of screening a test molecule for PKN antagonist activity comprising the steps of:

- a) contacting the molecule with a cancer cell;
- b) determining the level of PKN in the cell, thereby generating data for a test level; and

- c) comparing the test level to the PKN level of the cancer cell prior to contacting the test molecule, wherein a decrease in PKN in the test level indicates PKN antagonist activity of the test molecule.

18. (Original) The method of claim 17, wherein the level of PKN is determined by reverse transcription and polymerase chain reaction (RT-PCR).

19. (Original) The method of claim 17, wherein the level of PKN is determined by Northern hybridization or microarray analysis.

20. (Original) The method of claim 17, wherein the cell is obtained from a breast tissue, a colon tissue, an esophageal tissue, a bladder tissue, a brain tissue, a head and neck tissue, a kidney tissue, a liver tissue, a lymphoma tissue, a melanoma tissue, a pancreatic tissue, a lung tissue, an ovarian tissue, a prostate tissue, or a stomach tissue.

21-22 (Canceled).

23. (Original) A method of determining whether a test molecule has PKN antagonist activity, wherein the method comprises:

- a) determining the level of PKN in a test sample containing cancer cells, thereby generating data for a control level;
- b) contacting the molecule with the test sample to generate data for a test level; and
- c) comparing the control level to the test level, wherein no decrease in PKN in the test level as compared to the control level indicates that the test molecule has no PKN antagonist activity.

24 (Canceled).

25. (Currently amended) A method for determining the efficacy of a therapeutic treatment regimen in a patient, comprising:

- a) measuring **[[the]] at least one of** PKN gene copy number, **PKN mRNA, or PKN expression levels** in a first sample obtained from a patient, thereby generating an initial level;
- b) administering the treatment regimen to the patient;
- c) measuring **[[the]] at least one of** PKN gene copy number, **PKN mRNA, or PKN expression levels** in a second sample from the patient at a time following administration of the treatment regimen, thereby generating a test level; and
- d) comparing the initial and test levels, wherein a decrease in the gene copy number **[[level]], PKN mRNA, or PKN expression level,** in the test level relative to the initial level, **respectively,** indicates that the treatment regimen is effective in the patient.

26. (Original) The method according to claim 25, wherein the sample is obtained from a breast tissue, a colon tissue, an esophageal tissue, a bladder tissue, a brain tissue, a head and neck tissue, a kidney tissue, a liver tissue, a lymphoma tissue, a melanoma tissue, a pancreatic tissue, a lung tissue, an ovarian tissue, or a stomach tissue.

27-31 (Canceled).